Cytomegalovirus and Restenosis After Percutaneous Transluminal Coronary Angioplasty

To the Editor:

Manegold et al1 compared the incidence of prior cytomegalovirus (CMV) infection and the risk of restenosis after percutaneous transluminal coronary angioplasty (PTCA) in 92 consecutive patients. A total of 65% of these patients were positive for IgG CMV antibodies. At 6 months, angiographic restenosis was similar in CMV-positive and CMV-negative patients. These results conflict with those of Zhou et al,2 who identified prior CMV infection as an independent risk factor for restenosis after directional coronary atherectomy (DCA). Manegold et al explain this discrepancy on the basis of reduced vessel wall injury with PTCA in comparison with DCA.1 In the companion editorial, Bertrand and Bauters3 suggest that CMV may play a role in smooth muscle cell proliferation, which may be more important after DCA than after PTCA.

To address this question, we compared coronary restenotic tissue obtained by DCA from 44 patients who had restenosis after PTCA with tissue from 11 patients who had restenosis after DCA. Formalin-fixed, paraffin-embedded samples were stained by a trichrome method. Neointimal smooth muscle cell proliferative tissue was defined as loose connective tissue matrix containing numerous stellate cells, as quantified by computer-aided planimetry. Sclerotic tissue, fibrocellular tissue, atheroma, and thrombus were also quantified as previously described.4 Total plaque area was similar in both groups. The area occupied by neointimal smooth muscle cell tissue was greater in restenotic samples from patients with DCA restenosis (3.24±0.29 mm²) than in restenotic samples from patients with PTCA restenosis (1.28±0.22 mm²; P=0.0001). The percentage of total plaque area occupied by neointimal smooth muscle cell tissue was also greater in restenotic samples from patients with DCA restenosis (74±4%) than in restenotic samples from patients with PTCA restenosis (35±4%; P=0.0001).

This study documented an increased neointimal response in restenotic tissue after DCA in comparison with restenotic tissue after PTCA. The results of this study may help to understand why CMV—a promoter of smooth muscle cell proliferation and an established predictor for DCA restenosis—may not be a predictor for PTCA restenosis, which is predominantly mediated by a remodeling rather than a proliferative response.5

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